ANALYSIS OF FOCAL SEIZURES WITH CT CORRELATION

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CERTIFICATE

This is to certify that the dissertation titled **'ANALYSIS OF FOCAL SEIZURES WITH CT CORRELATION'** is a original work done by **DR.E.UMA MAHESWARI**, Post-graduate in General Medicine during the academic year 2005-2008. at Govt. Stanley Medical College Hospital, Chennai –1, which is to be submitted to The TamilNadu Dr. M.G.R. Medical University Chennai – 32 towards the partial fulfillment of the regulation for the award of **M.D. Degree in General Medicine** in March 2008.

Signature of the Unit Chief **Signature of the H.O.D of Medicine**

DECLARATION

I, **Dr.E. UMA MAHESWARI,** solemnly declare that dissertation titled, **'ANALYSIS OF FOCAL SEIZURES WITH CT CORRELATION'** is a bonafide work done by me at Govt. Stanley Medical College & Hospital during 2005-2008 under the guidance and supervision of my Unit Chief **Prof.Dr.T.VENKATAKRISHNAN, M.D.,**

The dissertation is submitted to The Tamilnadu, Dr.M.G.R.Medical University, towards partial fulfillment of requirement for the award of **M.D. Degree (Branch-I) in General Medicine .**

Place : Chennai **(Dr.E. UMA MAHESWARI)**

Date :

ABBREVIATIONS

1.	SPS	-	Simple partial Seizure
2.	CPC	-	Complex Partial Seizure
3.	SGS	-	Secondary generalised Seizure
4.	ATT	-	Anti - tuberculous treatment
5.	AED	-	Anti – Epiletic drugs
6.	PT	-	Pulmonary Tuberculosis
7.	DM	-	Diabetes Mellitus
8.	HT	-	Hypertension

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CONTENTS

INTRODUCTION

As early as 400 B.C. when Hippocrates wrote about epilepsy till the present day when medical sciences have reached the heights of sophistication with various investigations like Computed Tomography, Magnetic Resonance Imaging and Positron Emission Tomography, epilepsy – a common neurological disorder, remains an enigma.

Epilepsy is a clinical state, or syndrome, whereby a person is liable to recurrent epileptic seizures. Epilepsy may arise from a constitutional predisposition or as a consequence of acquired pathology and isolated seizures may be precipitated by a wide range of transient metabolic disturbances. A constitutional predisposition most commonly presents with so called primary generalized epilepsy whereas acquired pathology often produces focal epilepsy.

The physician's approach to a patient with epilepsy necessitates that he or she should first define the nature or type of the patient's seizures, then if possible, determine the site in the brain from which they are arising and finally specify the nature of the underlying pathology or pathophysiology. This idea, which cannot always be achieved in full, is particularly applicable to patients with focal epilepsy.

In the past, a large number of cases were labeled as epilepsy of unknown origin. Advances in imaging have revolutionized the ability to visualize the lesions in the brain that cause neurologic dysfunction. The introduction of computed tomographic scan (CT scan) particularly, has really helped to sort out the causes ofepilepsy. It easily picks up focal calcifications, mass lesions, vascular lesions, abscesses, ring or disc enhancing lesions and other various innumerable abnormalities. Thus, the CT scan, one of the modern noninvasive investigation is a valuable tool in detecting intracranial abnormalities in patients with focal seizures.

AIMS AND OBJECTIVES

1. To detect various CT-scan abnormalities in patients with focal seizures.

2. Study the value of CT-scan Brain in diagnosing the etiological factor of focal seizures.

3. Correlation between clinical features and CT scan findings.

REVIEW OF LITERATURE

The ability to identify the site of origin of a seizure on the basis of clinical manifestations began with the pioneering efforts of John Hughlings Jackson in the late 19th century which expanded with surgical studies conducted by Penfield and colleagues in the middle of the 20th century. It has further expanded in the last two decades with the increased availability of long-term video electroencephalographic recording. Jackson, an eminent British neurologist, defined epilepsy in the year 1837 as an intermittent derangement of the various systems presumably due to a sudden, excessive, disorderly discharge of cerebral neurons. Modern electro physiology offers no evidence to the contrary. Seizures are produced by an abnormal discharge from the cortical neurons resulting in stereotyped movements of the body, abnormal sensory perceptions or behavior. Epilepsy is the condition of recurrent seizures caused by an inherent abnormality of the brain.

Lord Russell gave a more detailed definition. According to him, "An epileptic seizure is defined as an intermittent stereotyped disturbance of consciousness, behaviour, emotion, motor function or sensation that on clinical grounds is believed to result from cortical neuronal discharge".

The basic physiological nature of epilepsy is expressed in Electroencephalographic terms (EEG). The spikes and sharp waves are the EEG hallmarks of inter-ictal recordings in patients with epilepsy. These are

due to hypersynchronisations of electrical activity within an abnormal pool of neurons.

Epilepsy is a symptom of many diseases and a genetic predisposition may produce seizures in many individuals and not in others. Also lesions of brain in Rolandic area and the anterior temporal lobe appear to have lower threshold for seizures. It remains undecided whether all seizures share a common mechanism in which threshold vary from person to person and in the same person from time to time are postulated to explain why some get seizures and others do not in the same situation.

Modern view of epileptogenesis concerns changes in neurotransmitter level as well as complex interactions within neurons at receptor sites on cell membrane. The superior region of the hippocampus is one of the most seizure prone area of the brain.

Hippocampal studies demonstrate that generation of the epileptogenic discharge depends on interplay between three factors:

1. The inherent capacity of certain normal neurons to elaborate active responses leading to sustained depolarization and paroxysmal bursting.

2. The breakdown of normal inhibitory mechanisms and the augmentation of excitatory synaptic mechanism thereby facilitating synchronous neuronal interactions. Postsynaptic GABA medicated

inhibition is suppressed and recurrent synaptic inhibition is sufficiently reduced to allow synchronization to develop.

3. The effects of modulation by neuro-transmitter substance help to trigger and maintain epileptic discharge. Acetylcholine mediates neuronal excitability and promote transmissions from an inter-ictal to an ictal state. Acetylcholine produces initial hyperpolarisation followed by prolonged depolarization. There is decreased conductance of Sodium and calcium currents so that any excitation to the affected neuron is favoured.

In a chronic epileptogenic focus,

1. Manifestations of excitability are subtle.
2. There is a high incidence of non-bursting cells.
3. The cellular voltages are low voltage and shorter duration.
4. Cellular interaction shows less synchrony.
5. Cell bursts correlate with surface spikes 50% of the time.
6. A large number of neurons are only partially or selectively affected; there are external inputs from the brainstem, thalamus etc., which are constantly changing.

7. There may be selective loss of GABA' ergic inter-neurons which leads to loss of effective post-synaptic inhibition.

8. Changes in neurological morphology could affect the density of different channels disturbing the balance between excitatory and inhibitory mechanisms.

9. Some ill-defined genetic factors may play a role.

Chronic foci show changes in neuronal morphology – loss of dendrites, simplification of the arborisation patterns, shrinking of the entire neuron, neuronal dropout and gliosis.

As epileptic discharges appear either when inhibition is decreased or excitation is increased, recent research has focused on the role of inhibitory neuro-transmitters like GABA and glutamate receptors exist. This may explain the heterogeneity of seizures and the differences in their response to treatment.

A particular type of receptor for the excitatory amino acids has been studied intensively - the N methyl D asparatate (NMDA) receptors which occur throughout the brain with the highest density in the hippocampus. The NMDA receptors mediate slow Excitatory Post-Synaptic Potentials (EPSP) which rise in time of order of 20-25 msec. and decays of 200-300 msec. These receptors are permeable to calcium but can change the generation of

inter-ictal epileptiform discharge. NMDA receptor antagonists have been shown to have anticonvulsant properties and are being studied for use as antiepileptic drugs. GABA' ergic neurons are preferentially lost at epileptic foci. While it appears certain that GABA plays an inhibitory role in epileptogenesis, its precise mode of action remains unclear.

Electrodes placed on the scalp may pick up high voltage, interictal spike discharges: which are a characteristic feature of epilepsy. Intra cellular micro-electrodes measures synaptic potentials that occur synchronously in several cells. These corresponds to the inter-ictal spikes recorded on the scalp are called Paroxysmal Depolarization Shifts (PDS) in resting membrane potential. The PDS are the basic building blocks from which the epileptogenic foci develop.

Neuronal membrane contains "voltage-gated channels", primarily responsive to the electrical field changes at the membrane and "transmitter regulated channels" regulated by neuro-transmitter receptors. Both voltage regulated and receptor-regulated channels are involved in the production of PDS. GABA is the major inhibitory neuro-transmitter in the CNS and is present in 30% of all synapses. Most GABA' ergic neurons form local inhibitory loops.

Nature of the discharging lesion

Seizure discharge can be initiated in an entirely normal cerebral cortex, as happens when the cortex is activated by administration of drugs; withdrawal of alcohol or other sedative or by repeated stimulation by sub-convulsive pulses (kindling phenomenon). The electrical properties of a cortical epileptogenic focus suggest that its neurons have an increased ionic permeability that renders them susceptible to activation by hypothermia, hypoxia, hypoglycemia, hypocalcemia & hyponatremia, repeated sensory stimulation and during some phases of sleep. Epileptic foci are characterized by spontaneous inter-ictal discharges during which neurons of the discharging focus exhibit large calcium mediated PDS followed by prolonged After Hyper Polarisation (AHP). The AHP are due to Calcium dependent K+ currents, better explained by enhanced synaptic inhibition. The PDS summate to produce surface recorded inter-ictal EEG spikes. The AHP correspond to slow wave of EEG spike and wave complex.

The neurons surrounding the epileptogenic focus are hyper polarized from the beginning and are GABA' ergic inhibitory neurons within the focus. Seizure spread depends on any factor or agent that activates the neurons within the focus or inhibits the surrounding neurons.

The level of extra cellular Potassium is elevated in glial scars near epileptic foci and a defect in voltage sensitive Calcium channels has been postulated. Deficiency of neuro-transmitter GABA, increased Glycine, decreased taurine, and increased glutamic acid etc. have been reported in human epileptogenic tissues.

Firing of epileptogenic neurons in cortical focus is reflected in EEG as a series of spike wave discharges that increase progressively in amplitude and frequency. Once the intensity of seizure discharge exceeds certain limit it overcomes the inhibitory influences of the surrounding neurons and spreads to neighboring cortical and sub-cortical synaptic connections.

If unchecked the cortical impulse spreads to the adjacent cortex and the lateral cortex via the inter-hemisphere pathways. The first clinical manifestation depend on that part of the brain from which the seizure originates. There is propogation to sub-cortical nuclei and spinal neurons via the corticospinal and reticulospinal pathways. The spread of excitation to the sub-cortical and brainstem centers correspond to the tonic phase of the seizure, loss of consciousness and signs of autonomic over activity. Soon after the spread of excitation, of diencephalo-cortical inhibition begins and intermittently interrupts the seizure discharge, changing it from tonic to clonic phase. The intermittent clonic burst become less and less frequent and eventually cease completely leaving in their wake exhausted neurons in the

epileptogenic focus and increased permeability of the blood brain barrier. These changes form the basis of Todd's post-epileptic paralysis. There is 2-3 fold increase in the glucose utilization of neurons during seizure discharge and the paralysis could be due to neuronal depletion of glucose and accumulation of lactate.

The spike and wave complex which represents brief excitation followed by slow wave inhibition is the type of pattern that characterizes the clonic (inhibitory) phase of focal motor or Grandmal seizures. Temporal lobe seizures arise in the medial temporal lobe, amygdaloid nuclei and hippocampus. They may also arise in the convexity of the temporal lobe. Loss of memory for events of the episode is due to the paralytic effect of the neuronal discharge in the hippocampus. The seizure focus may establish a persistent secondary focus in the corresponding control area of the opposite hemisphere via cortical connection called mirror focus. The mirror form is the source of confusion in trying to identify the side of the primary lesion by EEG.

Severe seizures may cause systemmic lactic acidosis with fall in arterial pH, reduction in arterial O_2 saturation and rise in PCO_2 which are secondary to respiratory spasm and excessive muscular activity. Heart rate, blood pressure and CSF pressure rise during seizure. The rise in BP evoked

by seizure causes significant increase in cerebral blood flow to meet the increased metabolic needs of the brain.

Imaging modalities used in diagnosis

At present the two most widely used imaging modalities in neuro-radiology are CT scan and MRI. CT is well suited for imaging of the bone, calcifications and hemorrhage. Hence, CT is still the mainstay in imaging, in the emergency room. Head trauma, suspected sub-arachnoid hemorrhages and fractures of the facial and temporal bones are examples. CT is better than MRI in detecting calcifications in certain lesions like certain tumors, granulomas and parasites. It is also very useful for those patients who cannot tolerate or have contraindications to MR scanning as in cases of claustrophobia patients, with metallic bone prostheses, intra-cranial aneurysm clips, pacemakers etc. CT is comparatively cheap and is available in most of the tertiary care centers.

A standard CT examination of the head consists of a series of contiguous tomographic sections usually 5-10mm thick from the posterior arch of atlas to the vertex. Most adult heads can be covered in 15-20 sections. Some units prefer to perform a helical scan of the head, which takes little more than 30 seconds, accepting a small reduction in image quality. In general, the modification consists of thinner section (1.5 – 3 mm), changes in

the plane of sections, magnification of areas of interest and the use of contrast media, usually intravenous or intrathecal.

Contrast media used for CT are:

A. Intravenous	:	Water soluble iodinated media.
B. Intrathecal	:	a. Water soluble iodinated media b. Gaseous : air or Xenon.
C. Inhalation	:	Xenon

Contra indications for intravenous contrast media are history of previous allergy, impaired renal functions, asthma, multiple myeloma and sickle cell anemia.

Indications for I.V contrast medium hinge on the assumption that abnormalities in the blood brain barrier (other than cerebral edema) will be present or that a hypervascular lesion will be found. Most primary and secondary intracranial tumors and AV malformations, particularly those over the tentorium, are visible without contrast media, but the significantly increased accuracy of diagnosis makes contrast enhanced scanning the method of choice. Infective or inflammatory processes, certain aneurysms and a small proportion of extracerebral collections may also benefit diagnostically from I.V contrast medium. The preferred CT technique in

stroke is without contrast medium although the latter may be given if perusal of the initial images suggest an alternative diagnosis. Ideally all patients should be scanned in the first instance without I.V. contrast medium so that the decision as to whether contrast medium should be used is based on the clinical and imaging findings in each case.

MRI is more sensitive than CT scan as an imaging modality in investigating seizure disorders and it is more specific too. MRI utilizes the magnetic properties of H^+ ions in tissues when subjected to a powerful magnetic field. MRI can be used to study CNS lesions like neoplasm, cerebral edema, demyelinations, degenerative disease and congenital anomalies. MRI is also routinely utilized for evaluation of those patients who are potential candidates for epilepsy surgery, eg. : Mesial temporal sclerosis. The contrast agent used in MRI scan is Gadolonium – DTPA. The use of MRI for evaluation of epilepsy in our country is not widespread because of its availability and high cost.

Positron Emission Tomography (PET) and Single Photon Emission Computerised Tomography (SPECT) are used to study cerebral regional blood flow, metabolic activity and glucose utilization. PET & SPECT can be used to determine the site of origin of seizures. In a recent study by Theodore et al, presence of glucose hypometabolism was found in the left temporal lobe of 70-80% of patients with temporal epilepsy, who have undergone surgery.

But even in affluent countries these investigative modalities are still largely used as research tools only.

CT scan can be used as the primary imaging modality in any case of partial seizures. If diagnostic dilemma remains or if surgical treatment is anticipated, MRI and other imaging studies may be advised. CT scan has better resolution for calcified lesions than MRI. Cerebral calcification can be due to metabolic, neoplastic, vascular, congenital or developmental, traumatic, infections or non-infections inflammatory causes, Metabolic processes like hyperparathyroidism and other abnormalities of calcium and phosphorous metabolism can cause bilateral basal ganglia calcification but these calcified foci do not themselves produce seizures by various mechanisms.

Tumors that are found during surgery for chronic epilepsy often lack radiological characteristics like edema, mass effect and contrast enhancement, that are usually typical of tumors. Examples are low-grade astrocytomas, oligodendrogliomas, gangliomas and more recently recognized dysembryoplastic neuroepithelial tumors. Calcification can be a manifestation of any of these tumors, especially on CT scans, but on MRI they usually appear as heterogenous masses rather than a uniform focus of calcification.

Vascular lesions that may give rise to epilepsy include infarcts, primary intracerebral hemorrhages and congenital vascular anomalies. The last category comprises arteriovenous malformations, venous angiomas, capillary telangiectasias and cavernous angiomas. Arterio-venous malformations consists of anomalous vessels that have the characteristics of both arteries and veins with intervening parenchymal tissue, and that are visible on MRI or conventional angiography; MRI shows characteristic multiple flow voids. Magnetic susceptibility studies are helpful in cases in which vascular lesions are suspected.

Venous angiomas have a linear or radial appearance and typically do not cause seizures or calcify. Capillary telangiectasias usually appear in the brainstem and do not cause seizures. Cavernous angiomas are present in about 0.5% of the general population and are frequent associated with epilepsy when they are symptomatic. The lesions are frequently multiple and usually enhance with contrast and the clinical course is much shorter.

Trematodes such as Schistosomes and Paragonimus, both of which are found in Asia and Africa, may infect the cerebrum, but typically also invade other organs such as the liver or lungs and usually cause seizures only in the acute stage of the diseases.

Cysticercosis is the other cerebral infection resulting in cerebral calcification and recurrent seizures, recent literature suggests that cysticercosis may be the most common cause of symptomatic epilepsy in the world [11,12,13]. Cerebral lesions usually evolve from active forms appear as thin-walled fluid filled cysts with mural nodule (the live scolex); it causes no inflammatory reaction. The transitional form is a more proteinaceous encapsulated cyst with ring enhancement; the cyst becomes a granulomatous, irregularly enhancing lesion as the organism dies. The inactive lesion contains the dead organism and is usually calcified, with no enhancement. At any of these stages there may be multiple lesions, most often the lesions are solitary. Seizures are the most common clinical manifestation at all stages of intra parenchymal infection, although focal headaches and focal symptoms are common during the active and transitional stages. Solitary areas of calcification are usually supratentorial and usually less than 1 cm in diameter without mass effect.

Classification of Seizures and Epilepsy syndromes

An epilepsy syndrome is a composite of signs and symptoms associated with certain pathologies or etiology (idiopathic). Many of the idiopathic syndromes have well defined prognosis.

An international classification of epileptic syndromes was proposed in 1981. The classification makes use of both clinical and EEG information. Classification of different epileptic syndromes based on seizure types occurring within the syndrome, age of onset and etiology will be of vital importance in the management of patients with epilepsy. The classification of seizures is constantly being modified. In the latest version (Epilepsia 30: 389, 1989); an attempt has been made to incorporate all of the epilepsies, epileptic syndrome and related seizure disorders and to categorise them not only as partial or generalized; but also according to the age of onset, primary or secondary nature of the seizure and the many clinical settings in which they occur. (Adapted from commission on the terminology and classification of International League against Epilepsy. Epilepsia: 26, 268-275 / 1985).

Classification of seizures and epilepsy syndromes

1. Localisation related epilepsies and syndromes

    a. Idiopathic with age related onset

        1. Benign childhood epilepsy with temporal spikes.

        2. Childhood epilepsy with occipital paroxysms.

    b. Symptomatic

Related to the area of onset and clinical EEG features (This encompasses most partial seizures).

2. Generalised epilepsies and syndromes

   a. Idiopathic with age related onset in order of age.

      1. Benign familial neonatal convulsions.
      2. Benign neonatal convulsions.
      3. Benign myoclonic epilepsy in childhood.
      4. Childhood absence epilepsy .
      5. Juvenile absence.
      6. Juvenile myoclonic epilepsy (impulsive petit mal)
      7. Epilepsy with Grand mal seizures (GTCS).

   b. Idiopathic and/ symptomatic in order of age

      1. West syndrome.
      2. Lennox Gestaut Syndrome.
      3. Epilepsy with myoclonic seizure.
      4. Epilepsy with myoclonic absence.

c. Symptomatic epileptic seizures as part of a clinical picture

AV malformation or degenerative diseases with or without metabolic etiology that present with seizures as part of the clinical picture.

3. Epilepsies and syndromes undetermined as to whether they are localized or generalized.

a. Both generalized and focal seizures

1. Neonatal seizures.

2. Severe myoclonic epilepsy in infancy.

3. Epilepsy with continuous spike waves during slow wave sleep.

4. Acquired epileptic aphasia (Landau Kleffner Syndrome)

b. Without unequivocal generalized or focal features

GTCS in which a focal or generalized onset cannot be determined by clinical or EEG features.

Special Syndromes

A. Situation related seizures

   a. Febrile convulsion.

   b. Seizures related to other identifiable situation such as stress, hormonal changes, drugs, alcohol or sleep deprivation.

B. Isolated, apparently unprovoked epileptic events

C. Epilepsies with specific modes of seizure precipitation. Eg. Reading epilepsy.

D. Chronic progressive epilepsia partialis continua of childhood.

Partial Seizures / Focal Seizures

All forms of seizures, possibly even the primary generalized type for which no cause is apparent are believed to originate in the cerebral cortex. Seizure beginning with aura at presentation may occur as generalized tonic and clonic seizures are considered to be of focal origin. Partial seizures vary in location of the lesion and are conventionally divided into two groups: Simple and Complex, depending on whether consciousness is retained or altered. Simple Partial Seizures most often arise from foci in the sensory or motor cortex. Complex Partial Seizures often have their origin in the temporal lobe on one side or the other. Types of seizures and localization of the offending lesions that give rise to them are listed below.

Common seizure patterns and their localization

CLINICAL TYPE	Localization
I. SOMATIC MOTOR	
Jacksonian (focal motor)	Contralateral pre-rolandic gyrus
Masticatory	Amygadaloid
Simple contra-versive	Frontal
Head and eye turning with arm movement.	Supplementary motor cortex
II. SOMATIC AND SPECIAL SENSORY MOTOR	
Somatosensory	Contralateral Posterolateral
Unformed images, light pattern	Occipital
Auditory	Heschels gyrus
Vertiginous	Superior temporal
Olfactory	Medial temporal
Gustatory	Insual
Visceral autonomic	Insular-orbital-frontal
III. COMPLEX PARTIAL SEIZURES	
Formed hallucinations, Illusions, Dyscognitive Experiences, Déjà vu, dreamy states, Depersonalization	Temporal cortex or Amygdaloid Hippocampal complex
Affective state (fear, depression or elation)	Temporal
Automatism (ictal and post ictal)	Temporal or frontal
Absence	Frontal cortex, Amygdaloid, Hippocampus.
Bilateral epileptic myoclonus	Reticulocortical

Source : Modified from Penfield and Jasper.

Classification of Partial seizures

A. Simple Partial Seizures (Consciousness not impaired)

1. With Motor Signs

    a. Focal motor without march.

    b. Focal motor with march (Jacksonian).

    c. Versive.

    d. Postural.

    e. Phonatory (vocalization or arrest of speech)

2. With somato-sensory or special sensory signs Simple hallucination

    a. Somato-sensory.

    b. Visual.

    c. Auditory.

    d. Olfactory.

    e. Gustatory.

    f. Vertiginous.

3. With Autonomic symptoms or signs (including epigastric sensation, pallor, sweating, flushing, piloerection and pupilary dialation.)

4. With psychic symptoms (disturbance of higher cerebral functions)

   These symptoms rarely occur without impairment of consciousness and are more commonly experienced as complex partial seizures.

   a. Dysphasic.

   b. Dynamic (eg. Déjà vu)

   c. Cognitive (dreamy states, distortion of time sense).

   d. Affective (fear, anger etc)

   e. Illusions (eg. Macropsia)

   f. Structural hallucinations (eg. Music, scenes).

B. **Complex Partial Seizures** (with impairment of consciousness; may sometime begin with simple symptomatology)

1. Simple Partial onset followed by impairment of consciousness

   a. With Simple partial seizures (A_1 – A_4) followed by impaired consciousness.

   b. With automatism.

2. With impairment of consciousness at onset

a. With impairment of consiousness at onset.

b. With automatism.

C. Partial seizures evolving to secondarily generalized seizures (may be generalized tonic – clonic, tonic or clonic)

1. Simple Partial Seizures (A) evolving to generalized seizures.
2. Complex Partial Seizures (B) evolving to generalized seizures.
3. Simple Partial Seizures (A) evolving to complex partial seizures and then to generalized seizures.

Classification of generalized seizures

Absence seizures

Typical absences

Impairment of consciousness only.

With mild clonic movements.

With atonic components.

With tonic components.

With automatisms.

With autonomic components (b through f may be seen alone or in combination)

Atypical absences may have

Changes of tone that are more pronounced than in A1.

Onset and / or cessation that are abrupt.

Myoclonic Seizures.

Clonic Seizures.

Tonic Seizures.

Tonic clonic seizures.

Atonic seizures (Astatic seizures)

Combinations of these may also occur eg., B&D, B&F, .. etc.,

Classification of status epilepticus

1. Convulsive status

A. Primary generalized major motor status

i. Tonic-clonic status

ii. Myoclonic status.

B. Partial or Focal status

i. Focal motor status (Jacksonian type)

ii. Epilepsia partialis continua.

C. Generalised major motor status with partial onset

i. Adversive (head, eyes or body onset)

ii. Partial motor onset.

2. Non-convulsive status

A. Petit-mal status

B. Psychomotor status or Complex partial status.

Risk Factor

Several studies have classified risk factors and prognosis for Generalized Tonic Clonic Seizures (GTCS), absence and complex partial seizures.

History of maternal convulsions, fever etc are risk factors for GTCS. History of febrile seizures was the only significant risk for absence seizures, 10% having had a simple febrile seizure and another 10% had atypical seizures.

History of epilepsy of febrile seizures in the mother, past history of febrile seizures or neonatal convulsions, cerebral palsy, head trauma and viral encephalitis were significantly more in patients with complex partial seizures than in control subjects.

Risk factors that were significant included twin pregnancy, breech presentation and perinatal asphyxia and prenatal / perinatal factors for complex partial seizures. Epidemiological studies indicate that parents and sibling of probands have more epilepsy than predicted population rates.

While about 4% of the population may have seizure of some type by the age of twenty, the rate for siblings and offspring of probands is near 10%. Comparable figures of epilepsy are 1% and 4%. Generic studies of patients with absence seizures suggest an autosomal inheritance for the seizure type.

The risk of recurrence of seizure is greatest in the first week after the initial seizure. 70% of the patients seen on the day of the first seizure have a second seizure by the second day; whereas for patients seen eight weeks after their first seizure, the risk of recurrence within the next three years is 22%. In other words, the longer a patients remains seizure free after an initial attack, the lower the likelihood of recurrence.

Ammon horn sclerosis or hippocampal sclerosis has been found in 50-60% of chronic epileptic patients. This abnormality consisting of severe neuronal loss and gliosis and tends to affect selective regions of the hippocampus .

In these cases atrophy is most obvious to the naked eyes. These pathological changes are unilateral in 80% of the patients. When sclerosis involves the Amygdala, the Uncus and spreads laterally to the

Parahippocampal gyrus and other parts of the temporal lobe, it is called Mesial Temporal Sclerosis.

Mesial Temporal Sclerosis is found in nearly 50% of the patients who undergo temporal lobectomy for intractable complex partial seizures. Changes have been demonstrated in the human brain after status epilepticus. Children suffer permanent brain damage more often and after a shorter duration of seizure than adults. Repeated seizures lead to ischemic cell damage in the hippocampal neocortex and cerebellum, Pathological changes are evident when seizures last for more than 90 minutes.

Ischemia, hypoglycemia and seizures are supposed to result in similar if not identical brain damage because all three results produce necrosis. However recent research has revealed fundamental differences in the mechanisms and types of brain damages produced by these conditions.

Tuberculoma:

Tuberculoma's of the brain account for 20-30% of intra-cranial tumors in India. In paediatric age group 41% of intra-cranial space occupying lesion have been found to be tuberculous in nature. Tuberculomas develop in the brain when the "Rich focus" does not rupture into the meninges but expands locally in the brain parenchyma. Tuberculoma may also originate from the meninges and superficial cortex. The meningeal form resembles a

meningioma. For reasons that are unclear the majority are supratentorial in adults whilst infratentorial granulomata predominate in children. In most patients lesions are solitary but in 15-34% of cases multiple tuberculomata are found (De Angelis 1981).[1]

Macroscopically a typical tuberculoma is a well defined grayish avascular mass with a yellow casting core. Histologically the central necrotic zone is surrounded by tuberculous granulation tissue containing epitheloid cells, lymphocytes and Langhans giant cells. Surrounding the tuberculoma there may be arteritis, neuronal damage and oedema of varying degrees of severity. As a tuberculoma regresses there is increasing collagen formation, sometimes associated with the deposition of calcium salts. Tubercle bacilli can be found in the majority of surgical or autopsy specimen, but in their absence, the pathological diagnosis can be made on the histological appearance of the granuloma alone.[2]

Intracranial tuberculomas occur mainly in adults in western countries whilst in India children are mostly affected (De Angelis, 1981).[1] Most intracranial tuberculomas present with symptoms and signs of a space occupying lesion and only a minority develop tuberculous meningitis in the course of illness. Compared to other intra cranial mass lesions, the incidence of convulsions is particularly high and in some series they occur in as many as 85% of cases (Arseni 1958).[20] Fever and general ill health are unusual

findings but a past history of tuberculosis or evidence of active infection outside the nervous system occurs in about 50% of the patients.[2]

60-70% of intracranial tuberculoma present with seizures. 56-93% have features of increased intracranial tension, 33-68% have focal neurological deficit. Gulati et al[3] found the commonest cause of focal seizures is tuberculoma. 63-73% of patients have single confluent large granuloma with necrotic centre. Tuberculomas may also be multiple [4].

Tuberculomas are avascular when studied angiographically, its appearance on CT & MRI varies. It is consistent with evolving phase of the disease. Initial phase of the disease – edema, necrosis may appear as low attenuating areas on CT scan. Once the granuloma starts to organize there may be high attenuation, contrast enhancement and calcification as well as ring enhancement with variable degree of surrounding edema. The enhancement may be homogenous or there may be a central radioluscent area corresponding to the central radioluscent area corresponding to a central zone of necrosis.

MRI are more sensitive than CT in detecting tuberculoma. Tuberculomas are isointense with grey matter on T_1 weighted MR images. On T_2 weighted images lesions show central hyperintensity. In some cases hypointense ring is present in the wall of a tuberculoma on T_2 weighted images.

A collar of high signal resulting from edema on T_2 weighted images. Tuberculomas enhance after IV gadalonium dimeglumine in a solid or ring paler. In tuberculoma a central speck of calcification target sign has been considered pathognomonic similar punctuate calcification may also be seen in cysticercous granuloma.[5, 6]

Presence of increased intracranial tuberculoma, Focal neurological deficit, along with CT features (> 20 mm size) irregular margin and mid-line shift were suggestive of intra-ctranial parenchymatous tuberculoma. [7] There will be evidence of pulmonary TB in 60% of cases of tuberculoma. [6]

Other causes for localized brain granulomatis lesions

Brain Abscess:

Central cystic lesion within a well defined enhancing ring lesion with substantial amount of edema. Tuberculous abscess may be clinically and radiologically indistinguishable from a pyogenic abscess.

Syphylitic gumma:

Solitary lesion in brain, but this lesion would be unusual without evidence of syphilis elsewhere.

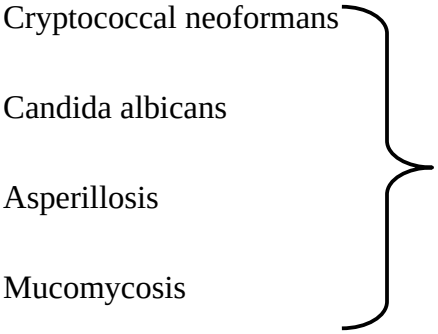
Nocardia :

Gram positive fungus behaves like a fungus than bacterium. Occurs in immunocompromised produces freely capsulated, frequently multiloculated liquefied abscess in brain.

Protozoal disease:

Produce focal brain lesions especially amoebiasis, toxoplasmosis. Acquired toxoplasmosis is a disease of immuno compromised.

Fungal disease - Intra-cranial granulomas



Usually causes chronic meningitis and results in solitary granuloma, multiple parenchymal brain abscess or granuloma. Candida granuloma located predominantly in white matter rather than cortex and associated with spinal fluid pleocytosis with evidence of candidiasis elsewhere in the body should be present.

Serological evidence of TB:

Based on recognition of serum IgG antibody to Mycobacterial antigen and the rise of ELISA. When the diagnosis is doubtful serological evidence of TB may prove useful. A positive test by ELISA technique can be taken as a supportive evidence of intracranial tuberculoma.[8] Major limiting factor for serological test is the cost.

TB elsewhere:

If facilities for serological studies are not available a reliable diagnosis can be made if there is evidence of TB elsewhere. [9] X-Ray should be taken for every patient. [1]

Newer methods:

1. Gene amplification by PCR to identify mycobacterial DNA.

2. If diagnosis of Tuberculoma is doubtful a trial of ATT may be instituted without histological confirmation.

3. Improvement in clinical and radiological features may provide a valuable evidence for diagnosis of lesions.

4. Response to ATT may not be rewarding due to the paradoxical enlargement of the lesion who are started on ATT.

CVA

A community based study of stroke showed an incidence of seizures in 4% of patients with cerebral infarction in 18% of patients with ICH and in 28% of patients with SAH.

It is estimated that cerebrovascular accidents account for 15% of all new cases of epilepsy and for more than 80% of new cases of epilepsy.

Neurocysticercosis

According to recent literature cysticercosis is the most common cause of symptomatic epilepsy in the world. The disease is cause by the larval forms of the tapeworm, Taenia solium and is endemic in much of Central America and South America, as well as Asia. It is associated with poor sanitation and is acquired through consumption of infected food or by feco-

oral transmission. The infected food may be pork but more commonly transmission occurs through consumption of contaminated fruits and vegetable grown in soil fertilized with pig or human waste.

Several thousand of stool samples of hospitalized patients in Northwest examined showed taeniasis in 0.52% and in slums and labor colonies, where pigs are raised the figure rose to 12-15%. Further 8-10% of slaughtered pigs harboured cysticerci.

T. Solium is the only tapeworm for which man can be both the intermediate host harboring the larval form of the worm, and the definite host harbouring the intestinal blood vessels into the venous circulation. They pass through the lungs and then embolise lodging in skeletal muscle, eyes and the CNS. In the CNS the oncospheres may lodge in the gray matter, at the junction of the gray and white matter, or in the subarachnoid space. In tissues the embryos develop into encapsulated larval forms called cysticerci which are filled with clear fluid and contain a visible scolex. When humans ingest contaminated meat, the cysticerci may attatch to the intestinal mucous and develop into adult worms 2-8 in length. The worms are composed of hundreds of proglottids, each of which contain the oncospheres which repeat the cycle when they are shed in the faeces. Autoinfection in cysticercosis can rarely occur when proglottids are regurgitated into stomach, which are digested by the gastric acid releasing the embryos.

The CNS is involved in 90% of cases of human cysticercosis. The presence of single cysticercosis in the CNS is unusual. In more than 80% of cases multiple cysts are found. Cerebral lesions typically evolve from an active to a transitional form and then to an inactive form.

Four types of CNS cysts are encountered in cysticercosis. Parenchymal cysts are usually found in the cerebral cortex, including the cortical-subcoritcal junction; the white matter is rarely involved. Meningeal cysts form in the meninges overlying the convex surface, sometimes causing hydrocephalus and stroke. Ventricular cysts are usually located in the 4^{th} ventricle and cause intermittent hydrocephalus and occasionally sudden death. Spinal cord cysts are rare.

The host can tolerate the worm as long as the embryo is alive. It unusually dies 2-6 years after infection and disintegration of the parasite eventually decays into grumose or eosinohilic dessicated materials. The final stage of this process is characterized by the presence of calcified nodule, presumably the result of dystrophic calcification of the necrotic larva.

Seizures are the most common clinical manifestation at all stages of intra parenchymal infestation, although headaches and focal symptoms are common during the active and transitional stages. Meningeal and intraventricular cysts can result in hydrocephalus.

The mechanism of development of seizures in neuricysticercosis is not known. One hypothesis is that the lesion disturbs the microenvironment of the surrounding neurons either by affecting neurotransmitters or by stimulating axonal reorganization by which they favour excitation or inhibition.

Diagnosis

The CT-Brain is useful in detecting small calcification. It is a good screening test. The main drawback are - posterior fossa lesion, lesion close to bone, lesions inside the ventricle and basal cisterns are missed out. The drawbacks of MRI Brain is that it may miss out small calcification but it is expensive and less available.

EITB-ENZYME LINKED IMMUNOELECTRO TRANSFER BLOT TEST. Done with lentin-lectin purified glycoproteins. The Serum is more sensitive than CSF. 100% specific and 98% sensitive. The main drawback are 30% of single intracranial lesion and calcified lesion may be tested falsely seronegative. CSF analysis is useful when unfractionated antigens are used

CLASSIFICATION

I. INTRAPARENCHYMAL CYST.

1. VESICULAR STAGE

The larval cyst actively evades the host's immune response through several mechanisms, inhibition of complement , cytokine release. So less inflammatory changes are seen in the surrounding tissue. Cyst has clear fluid. Parasite looks healthy and Viable cysticerci may remain alive for years.

2. COLLOIDAL STAGE.

Gradually host immune response overcome immune evasion mechanism. First stage of involution the vesicular fluid becomes turbid. Scolex show early signs of degeneration,.thick collagenous capsule develops. Surrounding brain parenchyma shows astrocytic gliotic changes with diffuse edema..

3. GRANULAR STAGE.

Cyst wall thickens . Scolex is transformed into coarse mineralised granule. Cysticercus is no longer viable.

4. CALCIFICATION.

Parasitic remanent appear as mineralised nodule. Oedema subsides. Astrocytic changes in the vicinity of lesion intensify. Exact duration of each stage is variable- depending on the individual endogenous immune response. Histopathological studies have shown cysticerci in different stages of involution in same individual, suggesting that host response is heterogenous.

IMAGING:

a) VIABLE CYSTS.

CT—hypodense, rounded cystic lesion, some may enhance contrast. MRI- cysts are hypointense inT1 and FLAIR., hyperintense in T2 sequence. MRI better than CT

b. DEGENERATING CYST

Degenerating cyst appear as contrast enhancing rings or nodules surrounded by areas of brain oedema. Scolex is not seen in CT/ MRI. FLAIR / DWI-MRI allow visualisation of scolex in some degenerating cysts. DD for single degenerating cyst is Tuberculoma.

c. CALCIFIED CYST

CT– hyperdense punctate dots. MRI shows areas of subtracted signals.

II. EXTRAPARENCHYMAL CYSTS.

Location: Convexity of cerebral hemisphere, Basal subarachnoid space, Sylvian fissure and Ventricle. Raised ICT is due to Mass effect, Distortion of normal anatomy of CSF pathway and direct obstruction to ventricular system by cysts. Inflammatory reaction in the meninges may lead to arachnoiditis.

III. VENTRICULAR CYSTS

Depends on involved ventricle and it is severe if the fourth ventricle is occupied. Contrast enhanced MRI is the choice to rule out the existence of viable cyst. Inflammatory reaction surrounds the cysts following cysticidal drugs and can cause acute hydrocephalus. Neuro endoscopic excision is promising alternative for cysticerci in lateral or third ventricle. If ependymitis is present – surgery to be preceded by shunt surgery.

IV. BASAL SUBARACHNOID /SYLVIAN FISSURE NEURICYSTICERCOSIS

Outside brain parenchyma cyst grow irregularly, and trigger more severe inflammatory response. Giant cysts develop in areas where more space is available - sylvian fissure. Worst prognosis associated with subarachnoid NC : Less common manifestations - basal meningitis, ventriculitis, angiitis, hydrocephalus. MRI is more accurate in diagnosis. Cerebral infarcts may

occur with cysticercotic arachnoiditis. Hydrocephalus* with intracranial cyst – cysticidal drugs to be used only after ventricular shunt has been placed.

DIAGNOSTIC CRITERIA

ABSOLUTE CRITERIA

- Demonstration of cysticerci by biopsy.
- Visualisation of the parasite in the eye by funduscopy..
- Imaging-cystic lesion showing scolex.

MAJOR CRITERIA

- Neuroradiological lesions suggestive of NC
- EITB-demonstration of antibodies in serum
- Resolution of cystic lesion spontaneously or after cysticidal drugs.

MINOR CRITERIA

- Lesions compatible with NC detected by imaging.
- Clinical manifestation suggestive of NC
- Demonstration of antibodies in CSF – ELISA method.
- Evidence of cysticercosis outside CNS.-cigar shaped soft tissue calcification.

EPIDEMIOLOGIC CRITERIA

- Residence in endemic area.
- Frequent travel to endemic area.
- House hold contact with an individual infected with T.solium.

DEGREE OF CERTAINITY

DEFINITIVE DIAGNOSIS

- 1 absolute criteria
- 2 major + 1 minor +1 epidemiologic criteria.

PROBABLE DIAGNOSIS

- 1 major + 2 minor
- 1 major + 1 minor + 1 epidemiologic criteria
- 3 minor + 1 epidemiologic

TREATMENT OF EXTRAPARENCHYMAL NC

Subarachnoid NC or Giant cyst	-	Albendazole for more than 1 month + high dose steroid or surgical excision.

Ventricular cysts	-	endoscopic aspiration, surgical resection and role of antiparasitic drugs not well established.
Hydrocephalus	-	no antiparasitic drugs, shunt surgery.
Arachnoiditis / Angiitis	-	no antiparasitic drugs . High dose steroids for more than 1 month
Ependymitis	-	no antiparasitic drugs . Shunt + high dose steroid

Other forms

Ocular and spinal cysts – only surgery.

STEROIDS

The Main form of therapy in cysticercotic encephalitis, angiitis and chronic meningitis is Steroids. Steroids ameliorate the secondary effects of headache , vomiting associated with cysticidal drugs.such manifestations are not due to the toxic effects of drugs but due to destruction of parasite in brain.

Patients with giant subarachnoid cysticerci , ventricular cysts , multiple parenchymal brain cysts – steroid to be given before , during and after the course of cysticidal drugs to avoid risk of cerebral infarcts , acute hydrocephalus , massive brain edema.

EITB :

Requires much more reagents, advanced equipments, highly trained personnel for antigen purification than ELISA. When available immunoblot is an excellent instrument for diagnosis and management.

CSF – ELISA :

The sensitivity of CSF ELISA is 87% and specificity 95%. Local synthesis of specific antibodies within the Central Nervous System seems to be the reason for the accuracy of ELISA in detecting brain cysticercosis through CSF analysis. In active forms of Neuricysticercosis showed no difference by ELISA with control patients. This finding is significant as it demonstrates the importance of selecting active forms of disease for medical management and helps to differentiate active disease from its remnant sequelae.[10]

False positive results are obtained when ELISA is done on serum samples due to cross reaction with E.granuloma which is a common cestode

infection. For this reason, it is much more useful when applied to CSF samples than serum.

Rajashekaran et al[7] has set up a diagnostic criteria to differentiate neuricysticercosis from tuberculoma based on imaging. The distinction is important because a single neuricysticercous granuloma is a benign condition whereas, tuberculoma is an active infection which requires prolonged treatment with toxic drugs.

Cysticerci lesions are usually round in shape less than 20mm in size with regular margins, visible enhancement or with visible scolex, without perilesional edema, whereas tuberculomas are irregular solid more than 20mm in size with perilesional edema and focal neurological deficit.

TUMOURS

Intracranial tumours are yet another cause of focal seizures. Apart from seizures, they also may present with headache, progressive decline in cognitive abilities or focal neurologic symptoms reflecting the particular anatomic site of the tumour.

When the physician examines the patient with suspected brain tumour, he or she should make a diligent search to look for primary malignancy elsewhere in the body. Then the neurologic examination of the patient should focus first on an evaluation of the mental status. The examiner should look

for evidence of specific localizing cognitive deficits, in addition to gleaning a sense of any personality change which has occurred. The patient is examined for increased intracranial pressure and cranial nerve abnormalities. Asymmetries of strength, sensation, visual fields and reflex activity should be sought. Attention should be paid to the constellation of signs suggestive of tumours in specific supratentorial, diencephalic or posterior fossa sites.

Advances in neuroradiology have contributed greatly to the diagnosis and management of patients with neoplastic diseases of the central nervous system. The virtues of CT scan remain those of speed, cost and the delineation of bone density. Contrast-enhanced CT imaging delineates intracranial masses as small as 0.5 cm in diameter. Tumours commonly appear as homogenous or ring-enhancing masses surrounded by variable amounts of edema. Although not a substitute for biopsy diagnosis, the CT scan often correctly predicts the histology of the tumour.

Nowadays, MRI (Magnetic Resonance Imaging) has been suggested as the optimal screening technique for the detection of most brain abnormalities. MRI using T2-weighted pulsing techniques appears to be even more sensitive than CT in the detection of focal structural abnormalities of the brain. Nevertheless, MRI has some disadvantages in comparison with CT for assessment of intracranial abnormalities. Cortical bone and intralesional calcifications emit no signal on MR images and thus areas of hyperostosis or

bone destruction, as well as tumour calcification are often not seen on MR images. These limitations, when considered with the higher cost and limited availability of MR equipment, suggest that CT will remain a primary imaging technique for the detection and localization of intracranial lesions for some time to come.[11]

MATERIALS AND METHODS

Thirty five patients with focal seizures who were admitted in the Neurology ward and General medical wards of Stanley Medical College Government Hospital during the period of Oct. 2006 to Oct. 2007, were enrolled for the study and was followed up.

Patients with simple partial seizures, complex partial seizures and secondary generalized seizures were included in the study. Patients with recent head-injury, doubtful history, mental retardation and children below 12 yrs were not included in this study.

A detailed history was recorded in each case (from the patient and intelligent attenders) regarding the age, number of episodes of seizures and parts of the body involved in the seizure have been recorded. Total duration of the seizure with shortest and longest interval between attacks and time of occurrence of fits in relationship with food and lunar cycles were also recorded. Important past history like ear discharge, psychosis, ECT, congenital, ischemic valvular heart disease, malignancy, exposure to STD, Pet rearing etc., were elicited and recorded. Dietary history regarding vegetarian and non-vegetarian was also taken. A thorough clinical examination was performed to detect any abnormality in CNS and screen for systemmic diseases with special attention to TB and cysticercosis.

Once a clinical diagnosis of focal seizure was made, patients were subjected to relevant investigations, besides the routine investigations. Focal seizures due to Metabolic cause was ruled out. Both Plain CT-scan and Contrast enhanced CT-scan of brain was done in patients to look for any abnormalities which could have caused the seizure. In CT-scan structural abnormalities like calcification, granulomatous lesions, tumors, vascular anomalies, infarcts, bleed, infections and inflammations was studied and the pattern of contrast enhancement was also studied. Follow up CT scan was done in cases which showed curable lesion like TB and Cysticercosis and the changes noted.

EEG was recorded in patients with normal CT scan or obvious metabolic abnormality to account for the seizure. A chest X-Ray was done in all cases to look for any supporting evidence of tuberculosis.

All patients were started on anticonvulsant medications and specific treatments were given for different disorders after establishing the diagnosis. Patients in whom no specific etiology could be identified received only Anti epileptic drugs and they underwent a repeat CT-scan after a period of 12 – 16 weeks duration.

OBSERVATION

Table 1 :

AGE AND SEX DISTRIBUTION OF PATIENTS WITH FOCAL SEIZURES

S. No	Age group	Male	Female	Total	%
1.	11 -20	4	4	8	22.85
2.	21 – 30	2	8	10	28.51
3.	31 – 40	1	11	12	34.28
4.	41 – 50	2	1	3	8.51
5.	51 – 60	-	-	-	-
6.	61-70	-	2	2	5.71
	TOTAL :	**9**	**26**	**35**	**100.00**

Table 2 :

TYPES OF FOCAL SEIZURES

S. No	Types of focal seizures	No of cases	%
1.	Simple Partial Seizures	11	31.42
2.	Complex Partial Seizures	4	11.42
3.	Secondary Generalised Seizures	20	57.14
	TOTAL	**35**	**100.00**

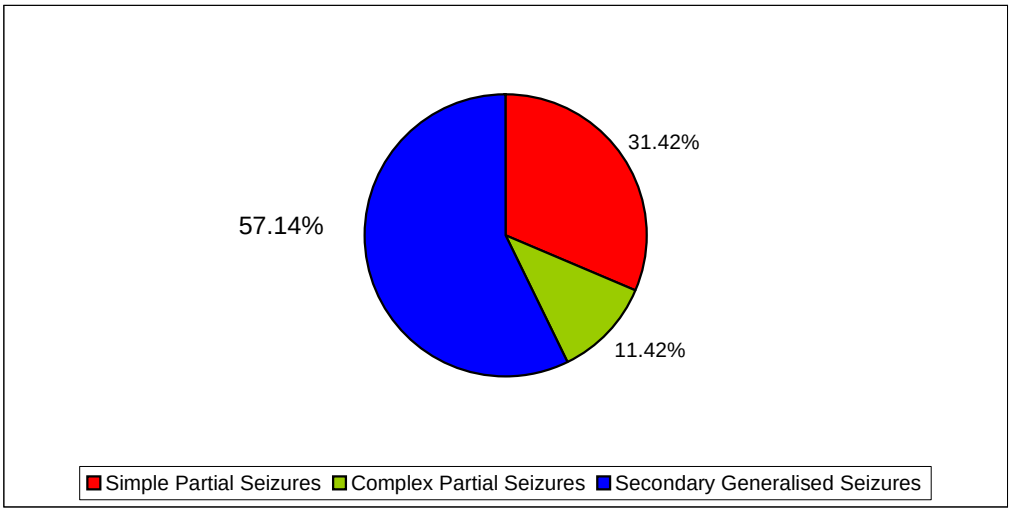


Table 3 :

Results of CT – Scan (brain)

S. No	Results of CT – scan	No of cases	%
1.	Normal	7	20
2.	Abnormal	28	80
TOTAL		**35**	**100.00**

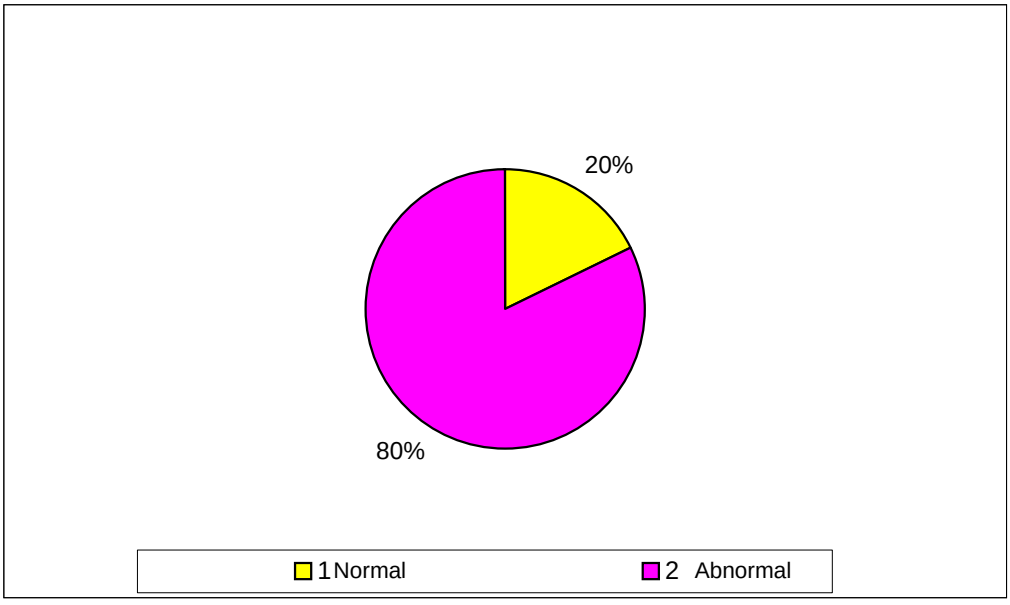


Table 4 :

Etiological diagnosis

S. No	Aetiology	No of cases	%
1.	Tuberculoma	12	34.28
2.	Neurocysticercosis	5	14.28
3.	Idiopathic	7	20.0
4.	Infarct	4	11.42
5.	Haemorrhage	3	8.57
6.	Multiple Calcification	3	8.57
7.	Secondaries	1	2.85
TOTAL		**35**	**100.00**

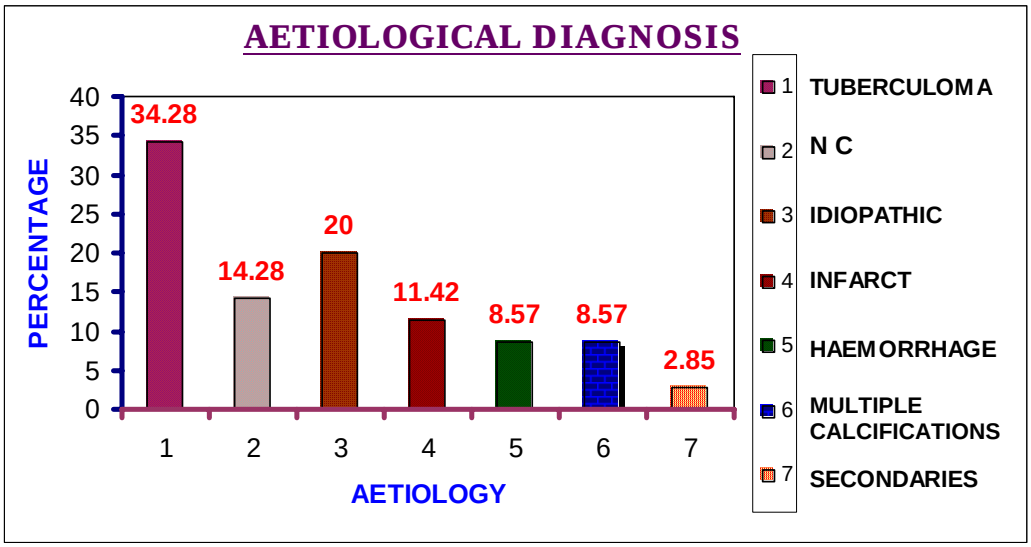


Table 5 :

Location of the lesion in CT - scan (brain)			
S. No	**Site**	**No of cases**	**%**
1.	Parietal	10	35.71
2.	Frontal	8	28.57
3.	Frontoparietal	1	3.57
4.	Parieto occipital	3	10.7
5.	Generalised	6	21.42
	TOTAL	**28**	**100.00**

Table 6 :

S. No	**CT – scan abnormalities in Neuricysticercosis**	
1.	Single Hypodense lesion with central scolex.	2
2.	Multiple small regular hypodense lesions < 2cms. In diameter	3
	TOTAL	**5**

Table 7 :

S. No	Investigations	
1.	CT- scan – typical target lesions with perilesional edema and contrast enhancement.	9
2.	Sputum AFP	2
3.	Mx	5
4.	CXR : PAView	2
5.	Lymph Node Biopsy	1

Diagnosis of Tuberculoma

DISCUSSION

Thirty five patients with focal seizures who were admitted in Govt.Stanley Medical College Hospital were studied. On analysis it was found that eight patients were below the age of 20 yrs, the youngest being 13 yrs and the oldest being 65 yrs. There was a male to female ratio of 3:1. The highest incidence of focal seizures was from the 31-40 age group.

The commonest clinical presentation of focal seizures was secondary generalized seizures (57.14%). In Verma[12] series he reported that majorities of his cases had secondary generalized seizures.

Of the patients registered 10 patients had lesions in the parietal lobe. Out of the 10 cases, 5 cases were due to tuberculoma. The commonest site of lesion was the parietal lobe. The next common being frontal lobe lesion as in the study of Kumar et al.[13] Of the 35 patients registered 28 patients (80%) had structural lesion and only 7 patients(20%) CT was normal. Japanese study done by Yagi et al, Ladumer et al showed that 30% of patients with focal seizures had normal CT.

There were 4 patients in our study who presented to us with partial complex seizures. In all the 4 of the cases the CT scan was normal. This is in contrast to the reported incidence of 40 – 70% abnormal CT scan of the brain,[8] in the patients with complex partial seizures.

In my study tuberculoma was found to be the commonest etiology of focal seizures (34.28 %), which goes according to the study done by Gulati et al.[3] Of the thirty five patients enrolled 12 patients had evidence of tuberculoma. 9 patients had large confluent solitary ring enhancing lesion. A study done by Grie et al[4] also showed solitary lesions common in tubercuoma. Of the 12 patients with tuberculoma 9 patients had the pathognomonic target lesion on CT-scan brain. In my study two patients had X-ray evidence of pulmonary tuberculosis whereas Adain et al[6] studies shows that 60% of cases had evidence of pulmonary tuberculosis on X-ray. Mx positive in five among 12 cases (41%) whereas study done by Vijayashekaran et al[14] showed tuberculin positivity to be 21% in case of Tuberculoma and 53% in case of lymph node tuberculosis.

The diagnosis of Tuberculoma was made in 12 cases using CXR, Lymph Node biopsy, Mx, sputum – AFB, CT scan of brain. 9 patients had the pathognomonic target lesion on CT scan of the brain[15], 2 patients has CXR evidence of TB and their sputum being positive for AFB and 1 patient had TB Lymphadenites. Tuberculin test was positive in only 5 cases among the 11 cases of tuberculoma (45-55%). Mx positivity is low in Central Nervous System TB when compared to other forms of TB. Tuberculoma – (21.2%) and Lympnode TB (53%).[14]

Three patients developed induration > 20 mm in diameter following tuberculin injection Kumar et al[16] diagnosed tuberculoma in 4 cases based on Mx. All the 12 patients were subjected to medical treatment and started with ATT for 9 months and they were followed up for nine months and a repeat CT scan brain was taken at the end of nine months. Also patients who had recurrent seizure in between were advised CT-scan brain whenever warranted.

Of the 12 cases of Tuberculoma, who were started on ATT 1 patient developed epilepsia partialis continua on day 4 of ATT and the other patient with complaints of recurrent seizures which required management in IMCU. A repeat CT scan taken showed an increase in the size of the lesion with perilesional edema. This is been described as a paradoxical reaction explained by Chambers et al[17] to be due to local tissue reactivity induced by altered meningeal permeability resulting in paradoxical worsening of lesions.

Neurocysticercosis was diagnosed in 5 patients. Though it is reported common in pork eaters[2], in this study all 5 patients were non-pork eaters. Desai[7] had reported a majority of the patients to be vegetarians in his study on Neurocysticercosis, but in our study all the patients were non-vegetarians.

The diagnosis of Neurocysticercosis was made in 2 patients who had the typical CT feature of a small cyst with hypodensity with well clear defined edges and a hypodense nodule inside the cyst showing a scolex

without perilesional edema or mid-line shift and also these patients did not have any focal Neurological defecit.[18]

Three other patients had small multiple cystic hypodense lesions less than 2cms. With regular margins without edema. These patients fulfilled Rajashekaran's et al criteria for diagnosis of Neuricysticercosis.[8]

Five patients was started on Albendazole and steroids for 4 weeks duration. Patient did not develop recurrent seizures or worsening of symptoms. A repeat CT was performed at the end of 4 months which show regression in the size of the lesion (3% of solitary cysticercous granuloma increase in size after starting Albendazole). In contrary none of our patients developed increase in size of lesion.

Four of our patients had cerebral infarction which is the 2nd most common cause of focal seizure and accounts 15% of all new case of epilepsy and haemorrhage accounted for 4% of seizures in elderly. The total incidence of ischemic stroke is 5 – 71%. Of the 7 patients with CVA, the patients were males with risk factors like HT / DM / smoking and develop focal neurological deficit following the seizure.

In our study only 2 patients above 60 yrs was registered of whom one had secondaries brain. Brain tumours are responsible for about 10 % of epilepsy. The incidence varies with age. In one Danish study 16.3 % of

patients who developed seizures after 25 yrs of age had brain tumour. In another study 14% of patients who developed late onset seizures after 60 yrs had brain tumours.

Three patients had calcified lesions in CT scan in which the possible etiology could not be made out. Theses patients were followed up and a repeat CT was performed at the end of 8^{th} week. Two of them showed a spontaneous dissolution of the lesion. 7 patients in a study had normal CT scan. EEG was taken for all these 7 patients and only three patients had abnormalities on EEG recordings. The rest 4 patients did not have any abnormalities on EEG.

Patients with focal seizures with a normal CT and a normal EEG who still have irretractable seizures should be further evaluated by MRI scan. MRI scan is more sensitive than CT scan in evaluation of epilepsy. Convers and Collegues performed MRI in 100 patients with irretractible partial seizures who had a normal CT and a normal EEG. 31 patients showed abnormalities on MRI of which 20 patients had temporal lobe epilepsy. MRI can detect hippocampal sclerosis which is the commonest cause of complex partial seizures.

However, MRI is not affordable by most of the patients in developing countries. The limited availability of MRI in Urban centres in India makes CT the initial investigation of choice of all patients with focal seizures.

SUMMARY AND CONCLUSION

1. 80% of patients with focal seizures showed structural lesions on CT scan.

2. The highest incidence of partial seizures was in 31-40 age group.

3. Male to Female ratio 3:1.

4. Tuberculoma of the brain is the commonest cause of Partial Seizures present in 31.42% of our patients.

5. The next common cause was Neuricysticercosis in 14.28% of patients.

6. The most common site of involvement of tuberculoma are parietal and frontal lobes and no infratentonal lesions were observed .

7. Since Neuro-imaging alone will not help in exactly delineating between Tuberculoma and Neurocysticercosis. Other corroborative clinical evidence must be taken into account while arriving at a conclusion.

8. Patients with Single Solitary Lesions on CT scan with no clinical / investigational back-up to diagnosis can be put on AED & wait and watch policy adopted and repeat a CT - scan after 12 - 17 weeks.

9. Though MRI is more specific and sensitive, given the socio-economic set up of our country, CT scan still remains to be the most valuable tool in making the etiological diagnosis of partial seizures.

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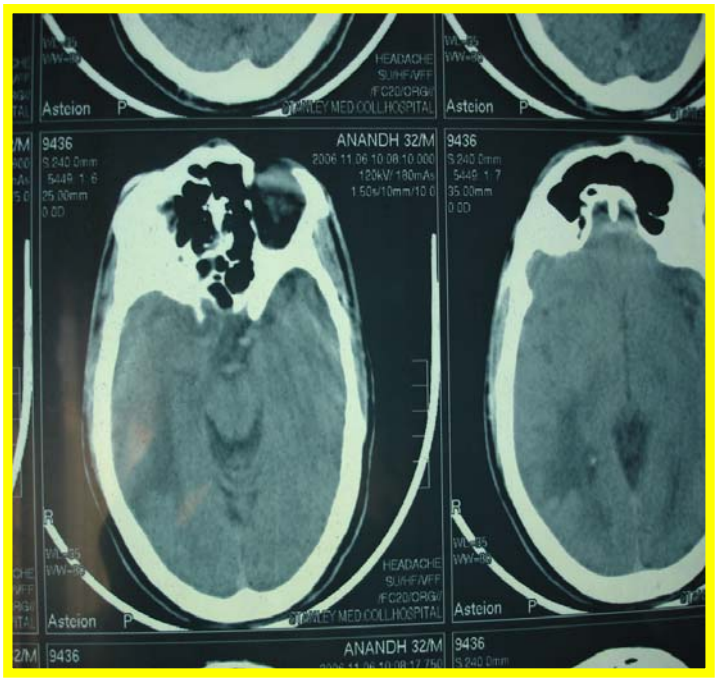
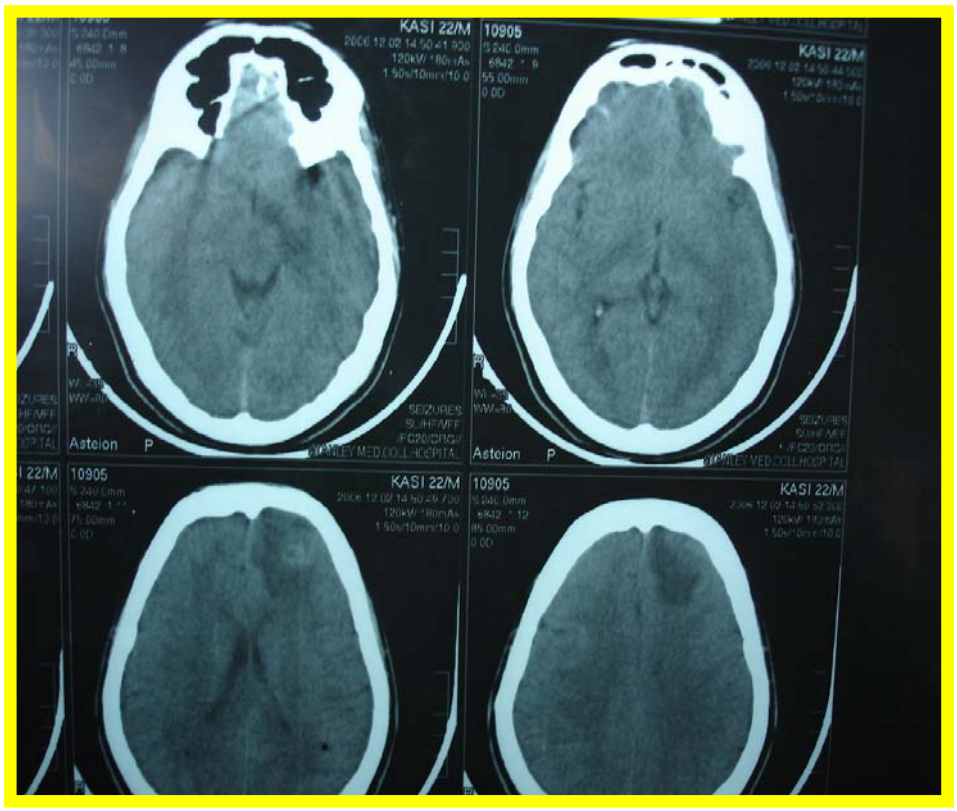


Plain CT Scan (brain) – hypodensity in Rt.Parieto –

Occipital region.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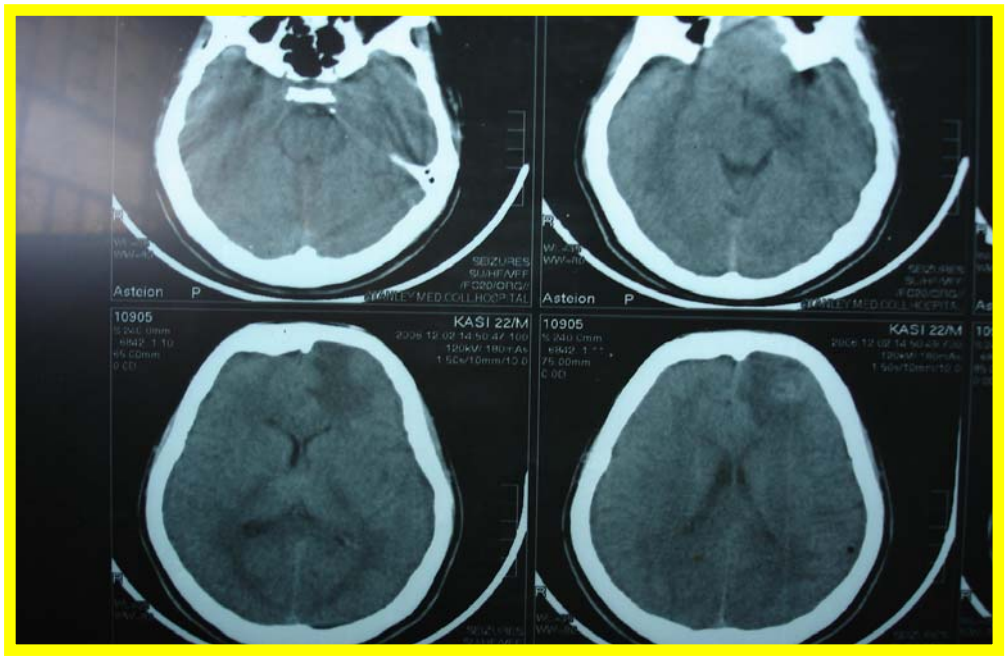
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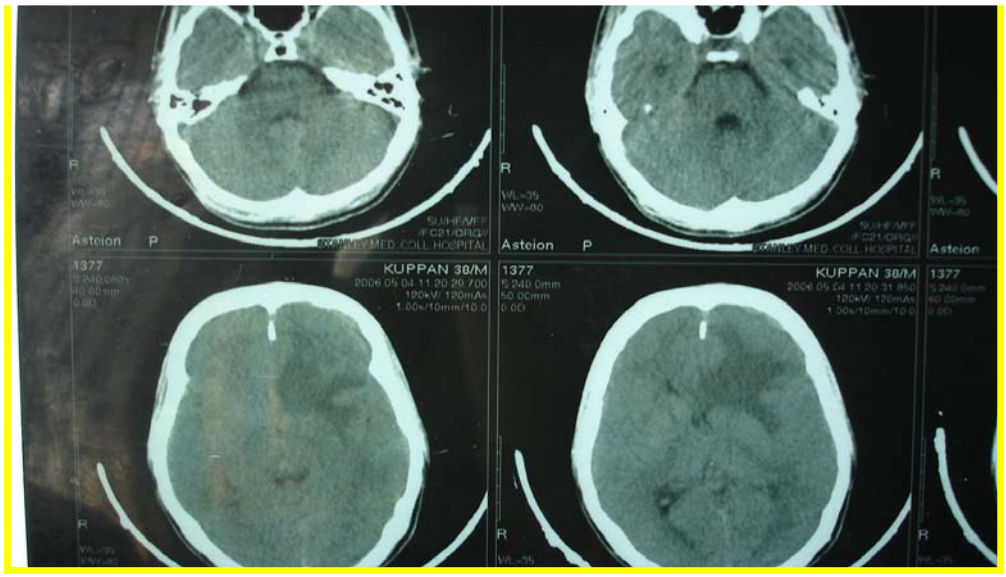
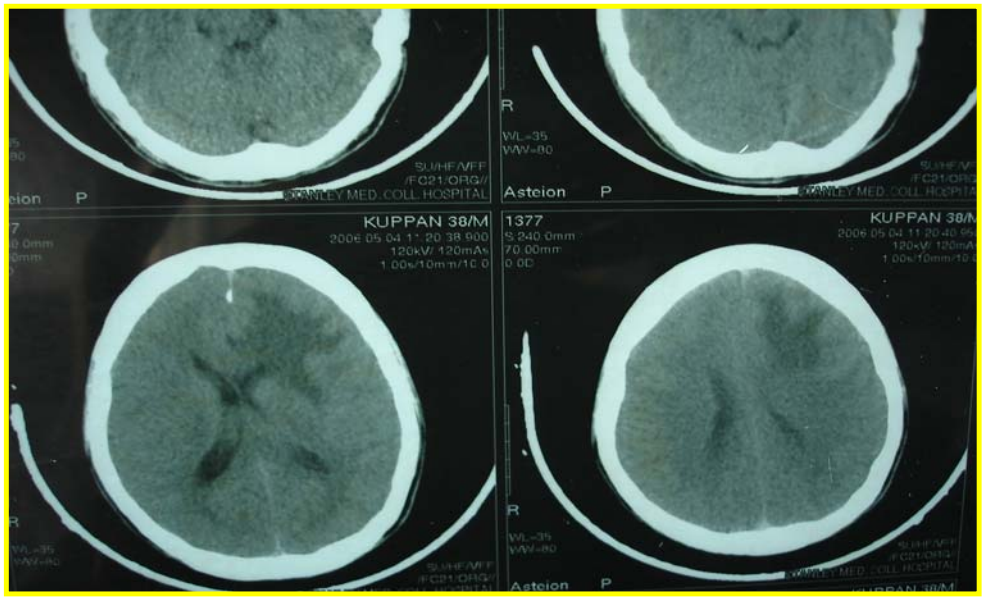
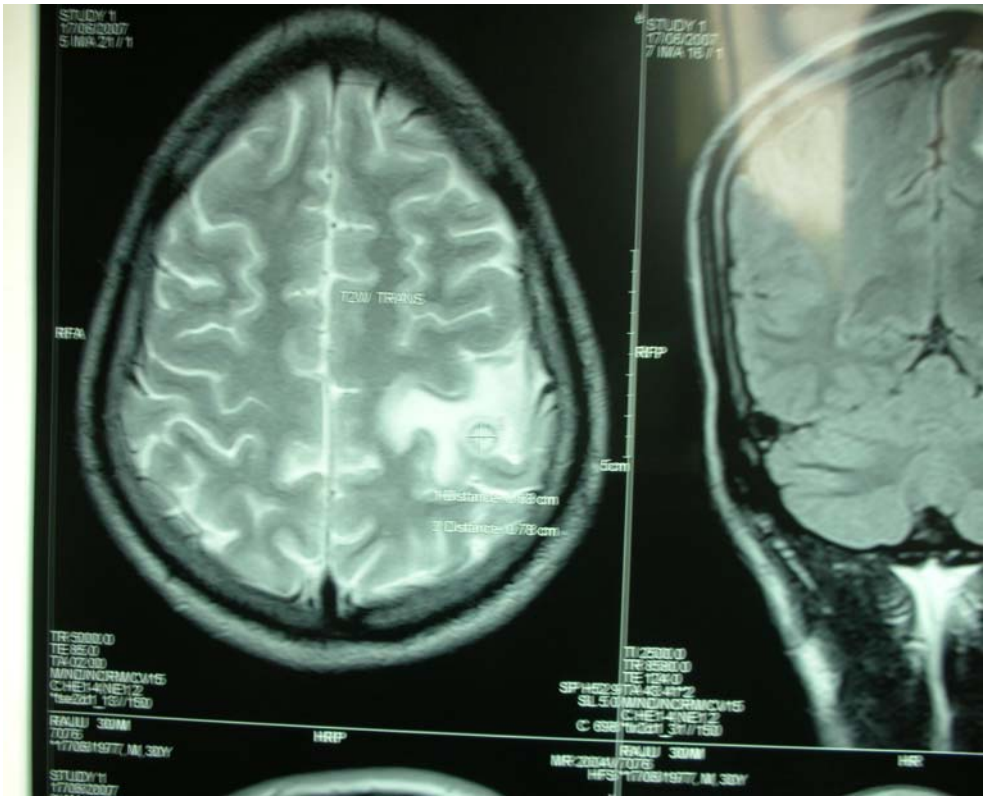
Plain CT Scan (brain) – hypodensity in left frontal region





Plain CT brain showing hypodense lesion with perilesional edema and mid – line shift.



Hypodense lesion with a scolex - Neuricysticercosis

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